WHAT IS CLAIMED IS:

1. A method for treating inflammation or immune disease in a subject comprising:

administering to the subject a pharmacologically active amount of a *Tripterygium wilfordii* Hook F root preparation, the preparation having anti-inflammatory and immunosuppressive pharmacological activity

wherein said method provides a steroid-sparing effect.

2. The method of claim 1 wherein the immune disease is an autoimmune disease.

3. The method of claim 2 wherein the autoimmune disease is rheumatoid arthritis, systemic lupus erythematosus or psoriasis.

4. The method of claim 1 wherein the preparation comprises triptolide.

5. The method of claim 1 wherein the pharmacologically active amount is about 60 mg/day.

6. The method of claim 1 wherein the steroid-sparing effect is further defined as providing pharmacological activity of the steroid at amounts less than capable of providing pharmacological activity in the absence of the *Tripterygium wilfordii*

Hook F root preparation.

7. The method of claim 6 wherein the steroid is prednisone.

8. The method of claim 1 wherein the preparation is an ethanol extract of *Tripterygium wilfordii* Hook F root.

9. A method of screening for a candidate substance having binding affinity for a glucocorticoid receptor comprising:

admixing a candidate substance with a glucocorticoid receptor in the presence of TwHF preparation or a glucocorticoid receptor binding component thereof; and

determining binding of the candidate substance to the glucocorticoid receptor.

10. The method of claim 9 wherein the glucocorticoid receptor is from a human skin fibroblast preparation.

11. The method of claim 9 wherein the glucocorticoid receptor binding component is triptolide, tripdiolide or wilforonide.

12. The method of claim 9 wherein the glucocorticoid receptor is conjugated to a label.

13. The method of claim 12 where the label is an enzymatic, chemical, or a radioactive label.

14. The method of claim 9 where the glucocorticoid receptor is conjugated to avidin/biotin.

15. A method of selecting a substance for treating inflammation or immune disease comprising:

admixing a candidate substance with a glucocorticoid receptor in the presence of TwHF preparation or a glucocorticoid receptor binding component thereof;

determining binding of the candidate substance to the glucocorticoid receptor;

selecting a candidate substance having binding affinity for the glucocorticoid receptor;

determining activity of the selected candidate substance for inducing steroid responsive gene expression; and

selecting the candidate substance being inactive for inducing steroid responsive gene expression.

16. The method of claim 15 wherein the glucocorticoid receptor binding component is triptolide, tripdiolide or wilforonide.

17. The method of claim 15 where the second determining step is carried out using a reporter gene construct under regulatory control of a steroid responsive element.

18. The method of claim 17 where the steroid responsive element is from the MMTV long terminal repeat.

19. The method of claim 17 where the reporter gene is the luciferase gene or the chloramphenicol acetyltransferase gene.

20. The method of claim 15 wherein the glucocorticoid receptor is from a human skin fibroblast preparation.

21. A method for inhibiting cyclooxygenase-2 induction in a subject comprising:

administering to the subject a pharmacologically active amount of a *Tripterygium wilfordii* Hook F root preparation, or a pharmacologically active component thereof, capable of binding glucocorticoid receptor.

22. The method of claim 21 wherein cyclooxygenase-1 activity is substantially unaffected.

23. The method of claim 21 wherein inhibiting cyclooxygenase-2 induction inhibits the synthesis of a prostaglandin, an autacoid, or a cytokine inhibited by

glucocorticoids.

24. The method of claim 21 wherein the preparation is a chloroform-methanol extract, a chloroform-ethanol extract, or an ethyl acetate extract of *Tripterygium wilfordii* Hook F root.

25. The method of claim 21 wherein the preparation comprises tripdiolide or triptolide.

26. The method of claim 21 wherein the preparation comprises wilforonide.

27. The method of claim 21 wherein the therapeutically effective amount is about 30-600 mg/day.

28. The method of claim 21 wherein the preparation has an LD_{50} in mice of greater than about 860 mg/kg.

29. The method of claim 21 wherein the preparation has an LD_{50} in mice of about 860 mg/kg to about 1300 mg/kg.

30. The method of claim 21 wherein the preparation has an LD_{50} in mice of about 1250 mg/kg.

31. The method of claim 21 wherein the preparation has a therapeutic activity:toxic index ratio greater than about $2.6x10^{-3}$.

32. The method of claim 21 wherein the preparation has a therapeutic activity:toxic index ratio from about $2.6x10^{-3}$ to about $4.5x10^{-3}$.

33. The method of claim 21 wherein the preparation has a therapeutic activity:toxic index ratio of about $4.5x10^{-3}$.

34. The method of claim 21 wherein the preparation has less than about 1.3 μg/mg triptolide.

35. The method of claim 34 wherein the preparation has from about 0.2 to about 1.3 μg/mg triptolide.

36. The method of claim 34 wherein the preparation has about 0.2 μg/mg triptolide.

37. The method of claim 21 wherein the preparation has an ID_{50} *in vitro* T-cell proliferation/LD_{50} ratio greater than about $2.6x10^{-3}$.

38. A method of blocking gamma interferon production in a subject comprising:

administering to the subject a pharmacologically active amount of a *Tripterygium wilfordii* Hook F root preparation, or a pharmacologically active component thereof, capable of blocking gamma interferon production.

39. A method of inhibiting interleukin-2 gene transcription in a subject comprising:

administering to the subject a pharmacologically active amount of a *Tripterygium wilfordii* Hook F root preparation, or a pharmacologically active component thereof, capable of inhibiting interleukin-2 gene transcription.